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MEDICAL ENTOMOLOGY.—*Experimental transmission of endemic typhus fever by the sticktight flea,* Echidnophaga gallinacea.[1]

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In 1931, Dyer, Rumreich, and Badger (1, 2) first demonstrated the natural infection of the rat fleas *Xenopsylla cheopis* and *Ceratophyllus fasciatus* with endemic typhus. The fleas were collected from wild rats trapped at typhus foci in Baltimore, Md., and Savannah, Ga. In 1931 and 1932 Dyer (3, 4) and collaborators were also able to demonstrate experimentally the susceptibility of these fleas to endemic typhus. In 1932, Mooser and Castaneda (5) reported experimental transmission of this disease by the following fleas: *Leptopsylla musculi*, *Ctenocephalus* (=*Ctenocephalides*) *felis*, *C. canis*, and *Pulex irritans*. Blanc and Baltazard (6) also reported *P. irritans* as a carrier of endemic typhus. In 1933 Workmann (7) reported the experimental transmission of endemic typhus by *Xenopsylla astia*.

The present paper deals with the experimental transmission of endemic typhus by the sticktight flea *Echidnophaga gallinacea*. So far as is known to the writer, the susceptibility of this flea to endemic typhus has not previously been reported.[2] The finding is of considerable interest in the Hawaiian Islands since the flea is of common occurrence on rats as well as on dogs, cats, mongooses, and chickens. According to a survey conducted by Eskey (8), *E. gallinacea* has been found on 13 percent of the rats trapped in the city of Honolulu. This flea frequently infests rats in large number, Eskey having shown that about 52 percent of the fleas collected on rats of the island of Oahu were found to be sticktight fleas.

EXPERIMENTAL DATA

On April 12, 1941, about 150 sticktight fleas were obtained from the ears of a dog in Honolulu. In order to be assured of absence of natural infection, 50 fleas were emulsified in physiological saline solution

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The writer is indebted to Drs. R. E. Dyer and N. H. Topping, of the National Institute of Health, U. S. Public Health Service, Washington, D. C., for supplying the Wilmington strain of endemic typhus used in the experiments reported in this paper. Acknowledgment is made also to Dr. R. D. Lillie, of the National Institute of Health, for the histological examination of the brain of one of the experimental animals.

[2] After this paper was sent to the editor, the writer noted a recent publication by Dr. G. D. Brigham (Pub. Health Rep. **56**(36): 1803–1804. Sept. 5, 1941) reporting the recovery of typhus virus from sticktight fleas (*E. gallinacea*) removed from two rats collected in Georgia. This report adds to the public health importance of these fleas.

and injected intraperitoneally into two guinea pigs. Neither of these animals developed any signs of typhus fever during a period of two weeks, nor was either found immune following experimental inoculation with a known endemic typhus virus (Wilmington strain) obtained from National Institute of Health, U. S. Public Health Service. The remaining fleas were placed on the body of a white rat freshly inoculated

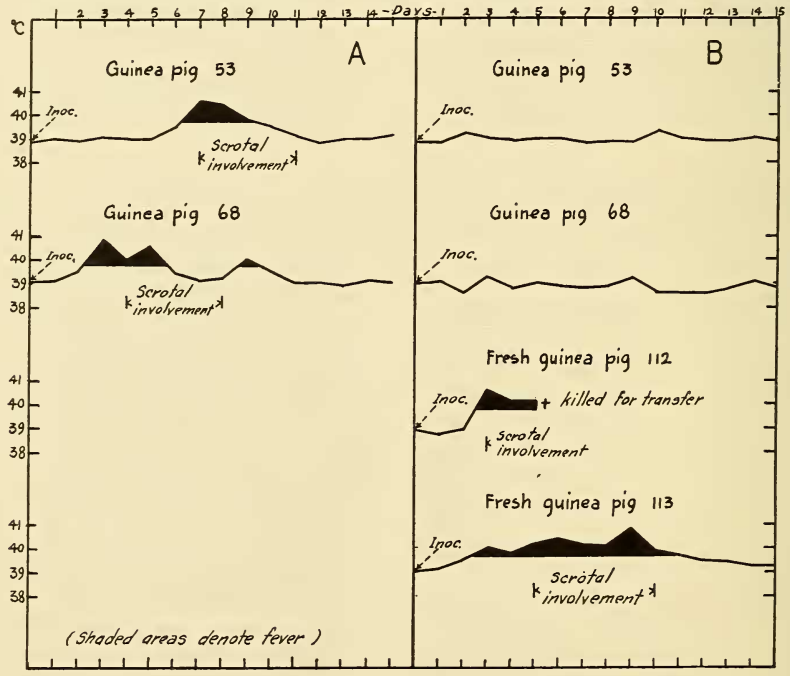

Fig. 1.—Cross immunity test: Daily temperature records of (A) guinea pigs inoculated with virus recovered from the sticktight fleas; (B) guinea pigs inoculated with the known endemic typhus virus (Wilmington strain).

with 2.5 cc of testicular washings from a guinea pig experimentally infected with the Wilmington strain of endemic typhus. Two guinea-pig controls, injected with the same inoculation, developed typical fever and scrotal reaction.

Most of the fleas that were placed on the rat attached themselves in a short time to various parts of the body particularly around the ears, eyes, and face. Thirteen days after the experimental infestation the rat was killed by a blow on the head, and 82 fleas were carefully removed. These fleas were placed into a small test tube overnight. The following morning the fleas and eggs were removed from the test tube, and all the feces of the fleas found adhering to the walls of the test tube were taken up in saline solution and inoculated intraperitoneally into a male guinea pig (No. 53). At the same time all the fleas were emulsified in saline solution and inoculated intraperitoneally into a male guinea pig (No. 54). A few days after these inoculations, both guinea pigs developed clinical typhus.

Guinea pig 53 was later found to be immune when inoculated with the Wilmington strain of endemic typhus (Fig. 1). Guinea pig 54 (first generation) was killed on the second day of fever and testicular involvement, and testicular washings from this animal were inoculated into guinea pig 57 (second generation). From this animal the strain was passed to three guinea pigs, 68, 69, and 70 (third generation), and later the strain was passed from guinea pig 69 to

guinea pig 97 (fourth generation). All the guinea pigs involved in the passage of the virus developed clinical endemic typhus. One of the animals (No. 68) of the third generation was then tested for susceptibility to endemic typhus and was found immune. Blood cultures made from the guinea pigs were uniformly negative. Scrapings from the tunica vaginalis of these animals also revealed intracellular rickettsial bodies.

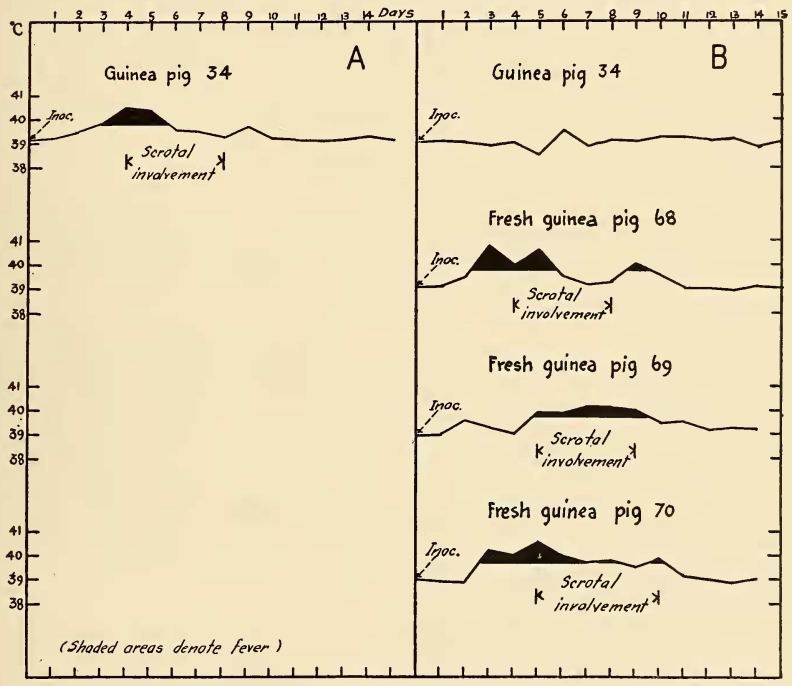

Fig. 2.—Cross immunity test: Daily temperature records of (A) guinea pig inoculated with the known endemic typhus virus (Wilmington strain); (B) guinea pigs inoculated with the virus recovered from the sticktight fleas.

As indicated in Figs. 1 and 2, cross-immunity tests were found to be complete between the flea strain of virus and that of the known endemic typhus (Wilmington strain).

A rabbit was inoculated with testicular washings from guinea pig 97 infested with the flea strain of virus. The rabbit was tested at weekly intervals for the presence of *B. proteus* OX_{19} agglutinins in the serum, and the following results were obtained: Just before inoculation and one week later, no agglutination in any dilution; second week, complete agglutination (four plus) in the 1:10 and 1:20 dilutions and partial (two plus) in the 1:40 dilution. Third week, complete agglutination in 1:10, partial (two plus) in 1:20, and traces (one plus) in 1:40. Fourth week, complete agglutination (three plus) in 1:10 and traces (one plus) in 1:20.

The brain of one of the guinea pigs (No. 70) reported in these experiments was submitted to Dr. R. D. Lillie, of the National Institute of Health, Washington, D. C., for histological examination. Sections from this brain revealed lesions which in type and distribution were consistent with typhus infection.

SUMMARY

The virus of endemic typhus (Wilmington strain) has been successfully transferred to sticktight fleas as a result of allowing the fleas to feed on an experimentally infected

rat. An emulsion of the feces of these fleas and an emulsion of the body of these fleas produced clinical typhus when inoculated into guinea pigs.

Clear cut cross-immunity has been shown in guinea pigs inoculated with the virus from the fleas and with a known endemic typhus virus (Wilmington strain).

Histological examination of the brain of one of the guinea pigs inoculated with the strain of virus recovered from the fleas revealed characteristic lesions of typhus fever.

Agglutinins for *B. proteus* OX_{19} were demonstrated in the serum of a rabbit inoculated with the strain of virus recovered from the fleas.

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ZOOLOGY.—*Description of a new genus and species of copepod parasitic in a shipworm.* Charles Branch Wilson, State Teachers College, Westfield, Mass.[1] (Communicated by Waldo L. Schmitt.)

So far as known, the first internal copepod parasites reported from the shipworm, *Teredo*, are some that were discovered by Dr. C. H. Edmondson, of the University of Hawaii, in the course of a study of shipworms taken from Honolulu Harbor. In view of the large number of Teredos that have been handled in the course of many studies of these destructive mollusks, the copepod parasite here described can not be very common or it would have been found before. Concerning its occurrence, Dr. Edmondson has written me as follows:

"The copepod was first observed during the fall of 1939, when fully 75 percent of the specimens of *Teredo milleri* Dall, Bartsch, and Rehder, 1938 (B. P. Bishop Mus. Bull. 153: 209, 210) over 30 mm in length recovered from Honolulu Harbor were found to be parasitized. The parasite has appeared in shipworms at three additional localities about Oahu, and also in Hilo Harbor, Hawaii, and at Kahului, Maui.

"Six shipworms, five species of *Teredo* and one of *Bankia*, in Hawaiian waters are known to serve as hosts of the parasite.

"The female clings tightly to the lining of the infrabranchial cavity of the host by means of stout, sharp mouthparts, while the male is likely to be unattached in the cavity and when released from the host is capable of swimming quite freely. Because of the greatly inflated body the female is capable of but slight movement when detached from the shipworm."

Teredicola, new genus[2]

Diagnosis.—Female: First three thoracic segments enlarged and fused with the head into a cylindrical body. Fourth and fifth segments abruptly reduced in length and width; genital segment about as large as the fifth segment; abdomen 3-segmented; caudal rami slender rods, each tipped with two setae. Ovisacs as long as the enlarged anterior body; eggs minute and numerous.

Male: Much smaller than the female, first segment only fused with the head, the others free. The first four segments with lateral plates diminishing in size backward. Abdomen 4-segmented, segments about equal in size. First antennae 6-segmented; second antennae 2-segmented, prehensile; maxilliped one stout

[1] Dr. Wilson completed this paper some months before his death on August 18, 1941. Received October 28, 1941.

[2] Dr. Wilson did not specify the family for this genus, but in correspondence with Dr. Edmondson he mentioned that it "evidently belongs to the family Clausidiidae which includes many of the Cyclopoida that infest annelids and mollusks."—W.L.S.